SENSORY AND MOTOR DISTURBANCES IN PARTS ABOVE THE DISTRIBUTION INVOLVED BY DEFINITE ORGANIC LESIONS OF THE SPINAL CORD.*

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The object of this paper is to call attention, first, to the gradual extension of the areas of disturbed sensation above the peripheral distribution related to definite lesions of the spinal cord, and secondly, to the marked increase in the tendon and skin reflexes in the same parts, and in parts above. These facts have not, heretofore, been noted.

These observations are based upon three cases, one with necropsy. In all, the lesions were of long standing, being 35, 29 and 4 years in duration, and the sensory phenomena were carefully studied and noted at varying intervals. The lesions were of traumatic origin, and the symptoms were of such clearness that it was possible in each case to make a clear-cut diagnosis in definite localities of the spinal cord.

The cases are as follows:

Case 1. Morgan. Was admitted to the nervous wards of the Philadelphia Hospital June 13, 1899, where he still is. His past and family history are of no importance, has never used alcohol or tobacco; and has never had syphilis.

When 27 years of age he was struck across the lumbar spine by a piece of iron and rendered unconscious. When he recovered, he was unable to use his lower limbs, and has never had more than slight use of them since. He has never had any bladder or rectal disturbances, or bed sores. Considerable pain was present in his lower limbs for some years after the injury, and girdle sense has always been present.

Notes by Dr. Van Epps made in January, 1900, state that the patient had from January 1 to 14 herpetic eruptions accompanied by much pain in the left side of the body extending from mid to mid line laterally and from costal margin to iliac crest posteriorly, and the pubic line anteriorly. The vesicles gradually disappeared but the pain continued for some time.

Examination on November, 1900, 28 years after the injury, by me, denoted almost total loss of power in the lower limbs. They were considerably wasted and spastic, more so the right. The cremasteric, patellar and plantar reflexes were present and prompter than normal; ankle clonus was present on each side, but the Babinski reflex could not be obtained.

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Sensation for all forms was absent to a line corresponding

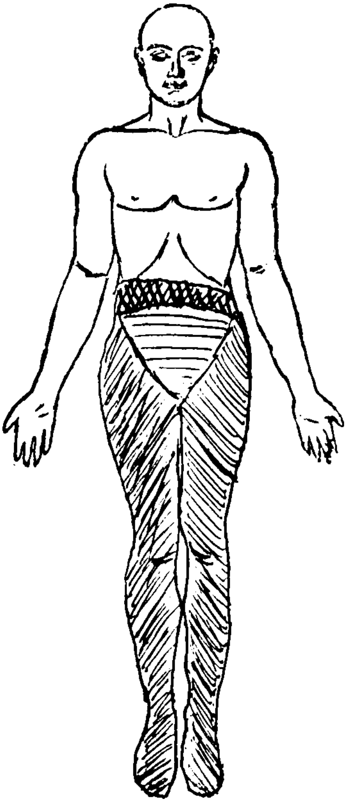

Fig. 1.

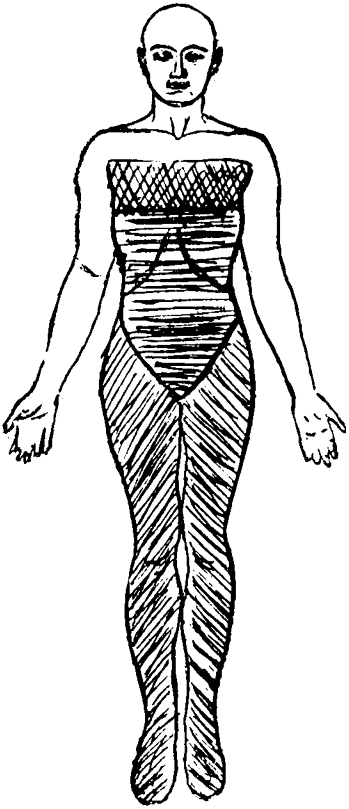

Fig. 2.

Fig. 1. The shaded areas represent disturbed sensation in 1900.
Fig. 2. The shaded areas represent disturbed sensation in 1907.

with the umbilicus. Above this point pin prick was appreciated, but touch and temperature sensations were not normally recognized until two inches above the umbilical line; just above this line there was a zone of hyperesthesia and the patient complained here of a girdle sense.

Examination by me on February, 1907, seven years after the last examination and thirty-five years after the injury, showed practically the same condition as to his motor power in the lower limbs. He could only slightly move the toes and knees. The cremasteric and patellar reflexes were still increased, but ankle clonus could not be obtained; the Achilles jerks were present. Plantar irritation produced flexion of all the toes. The bladder and rectum were normal.

The state of the sensation altered considerably. In my first examination of this year, he was unable to recognize touch, pain and temperature sensations over the lower limbs and abdomen up to the line of the nipples, and sensation did not really become normally appreciated until about the first intercostal space. Subsequent examinations have confirmed this, with the exception that pin prick was occasionally interpreted correctly below the nipple, but never more than two inches above the line of the umbilicus. (Fig. 2.) Bone sensation as tested by the tuning fork was diminished in the lower ribs, but was gradually better appreciated as the upper ribs were approached, and was normal about the second rib.

Power in the upper limbs was normal, and there was no apparent atrophy present. In the finger to nose test some ataxia was noticeable, but this was not marked. There was some hypotonia in the elbow joints. The sense of position was about normal, but on one occasion when absolute relaxation was obtained, it was demonstrated to some extent in the fingers, wrist and elbow joints. The biceps, triceps and wrist reflexes were prompter than normal, more so in the right side. On tapping with the percussion hammer over the chest and shoulder prompt contraction could be obtained in the corresponding muscles, so that practically reflexes could be obtained wherever desired.

X-ray examination made by Dr. Leonard D. Frescoln showed an inflammatory thickening (probably periostitis) around 10th to 12th thoracic and 1st lumbar vertebræ.

Summary. A man of 27 years, as a result of a severe blow to the 10th, 11th and 12th thoracic and first lumbar vertebræ sustained complete paralysis of the lower limbs. There must have been an almost complete transverse myelitis of the 9th, 10th, 11th and 12th thoracic spinal segments. An examination made by me 28 years after the injury denoted a spastic paralysis of the lower limbs with increased tendon reflexes, and disturbed sensation up to a line 2 inches above the umbilicus. The bladder and rectal functions were at no time involved. Examination made also by me 7 years later and 35 years after the injury showed the same condition as to motor power in his lower limbs, but sensation was disturbed to the first intercostal space. The tendon and skin reflexes in the upper limbs and in the reflex arcs of the chest and shoulder muscles were markedly exaggerated.

Case 2. Egan. Was admitted to the nervous wards of the Philadelphia General Hospital, May 11, 1892. His family and past history were of no importance and there was no history of alcoholism or syphilis. In 1884, at the age of 35, the patient lost his balance while on a running freight car, and fell to the track, striking his lumbar spine. He then lost almost all power in his lower limbs and control of his bladder and rectum.

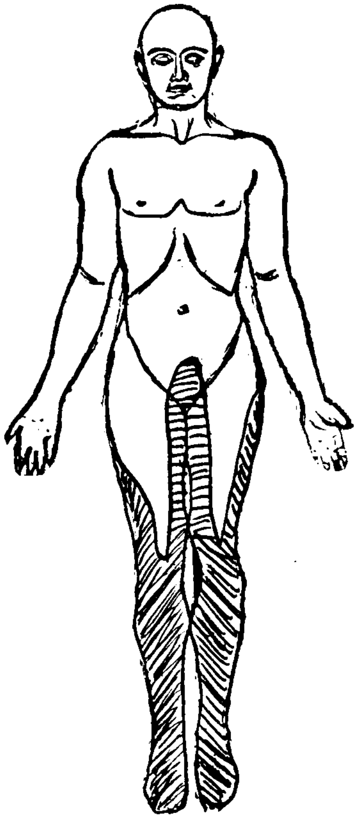

Fig. 3.

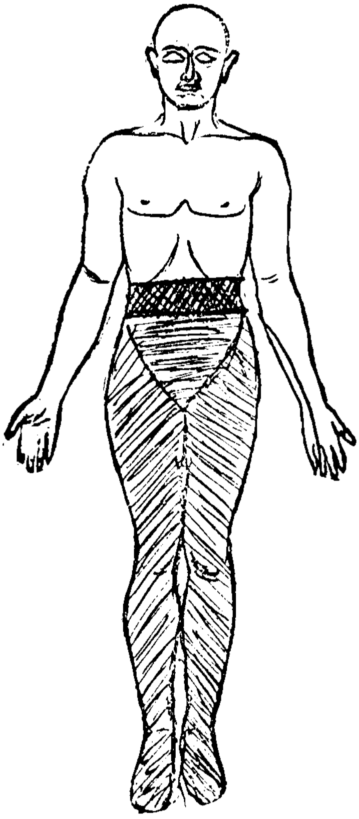

Fig. 4.

Fig. 3. The shaded areas represent disturbed sensation in 1899.
Fig. 4. The shaded areas represent disturbed sensation in 1907.

Notes made in June, 1899, 15 years after the injury, by Dr. G. E. Pfahler, state that the patient had loss of power in the lower limbs with the exception of some ability in flexing the right thigh on the abdomen. Bladder and rectal functions were completely lost. Sensation was lost as indicated in chart No. 3.

Examination by me in February of this year (1907), 23 years after the injury and eight years since the last examination, showed almost the same condition as regards the motor power of the lower limbs. The only movement possible was that of slight flexion of the right thigh on the abdomen. The toes were extremely hyperflexed and cyanotic. All of the tendon reflexes in the lower limbs were lost. Plantar irritation produced no movement of the toes. Bladder, rectal and sexual functions were totally lost.

Sensation for all forms was totally lost in the lower limbs and buttocks, with exception of a small area just in the inner surface of both thighs, and over the lower parts of the abdomen, to an irregular line about two inches below the umbilicus. From this line to a line drawn about 1 to 1½ inches above the umbilicus, sensation became gradually better appreciated, being normal at the above point. (Chart No. 4.)

Power and movement was normal in the upper limbs. The biceps and triceps tendon reflexes were very prompt, more so on the left side. On tapping with a percussion hammer over the chest and shoulders prompt contraction could be obtained in the corresponding reflex arcs. The abdominal reflexes were lost on both sides, as were also the cremasteric. In the finger to nose test, some ataxia was present, more so in the left limb. Sensation was normal in the upper limbs.

The X-ray examination denoted caries of the body of the third lumbar vertebra, especially towards the left side.

Summary. A man of 35, as a result of a fall, became completely paralyzed in the lower limbs with total loss of bladder and rectal functions. There must have been a complete transverse myelitis of all portions of the cord below the 2nd and 3rd lumbar segments. An examination made 15 years after the injury denoted almost a complete flaccid paralysis of the lower limbs with the exception of a slight ability to flex the right thigh on the abdomen. The bladder and rectal functions as well as all of the tendon reflexes were totally lost. Sensation was disturbed over both lower limbs, buttocks and perineum with the exception of a tringular area over the front of the thighs. An examination made 8 years later and 23 years after the injury showed the same condition as to the motor power in the lower limbs, but sensation is now disturbed to a line about 1½ inches above the umbilicus. The tendon and skin reflexes in the upper limbs and over the chest and shoulder are markedly increased. There is some ataxia in the upper limbs.

The third case, a brief history of which is only given, does not illustrate the sensory and motor changes which are the subject of this paper, but is used because it was a case of traumatic myelitis in which a careful microscopic examination was made, and in which the pathological findings are illustrative of the changes which probably have occurred in the two similar clinical cases here recorded.

Case 3. This case was recorded by Dr. Spiller and myself in the *Review of Neurology and Psychiatry,* October, 1904. A young man, because of an injury to the back, sustained complete paralysis of both lower limbs and of the bladder and rectum. All of the tendon reflexes of the lower limbs were lost. When examined by me one year after the injury, sensation was lost to a line corresponding about 2 inches below the umbilicus, and further examination made 3 years later and 4 years after the injury denoted absence of all sensation to a line drawn about 1 inch below the umbilicus.

A careful microscopic examination of sections taken from the upper part of each segment of the spinal cord from the 10th thoracic to the upper part of the cervical enlargement showed that as high as the upper part of the 10th thoracic segment, the cord was completely destroyed. Many normal fibers entered the cord in the posterior roots of the 9th thoracic segment.

Examination of the sections above the 9th thoracic segment stained by the Weigert hematoxylin method showed that the posterior root entrance zones, and the columns of Burdach and Goll were much better and more deeply stained than the other portions of the cord. A marked thickening was also found in the walls of the arteries, besides the usual ascending secondary degenerations.

We have here, therefore, two cases of definite lesions of the spinal cord; one involving the 9th to the 12th thoracic segments; and in the other the spinal cord below the 2nd or 3d lumbar segments. In both the lesions were of long duration, being 35 and 23 years, and the sensory symptoms were studied and noted at intervals of 7 and 8 years respectively. The areas of sensory disturbances increased in the period of 7 years in the first case from a line drawn 2 inches above the umbilicus to the 1st intercostal space, and in the second case from irregular areas over the thighs to a line drawn about 1½ inches above the umbilicus. In these areas of increased sensory phenomena, sensation was lost absolutely only about 2 or 3 inches, and above these parts there was a gradual shading off of disturbed sensation. The important point is that the areas of disturbed sensation have markedly increased above the limits previously found.

What are the causes of this increase in the sensory disturbances? The first answer which naturally presents itself, is an extension of the myelitic process, and secondly, the possible influence of the secondary ascending degenerations.

Let us consider first the possibilities of the occurrence of an ascending myelitis. It is well known that whenever the spinal cord is injured, even in a restricted portion, that the parts both above and below the point of injury may have small areas of hemorrhage or of softening. In fact the whole extent of the spinal cord may be so involved. In most instances after the subsidence of the acute process, these areas of softening and myelitis disappear and no trace of them is to be found, and clinically no symptoms appear. It is more than probable, however, that they are replaced by neurogliar tissue and we may have here the origin of a future sclerotic process.

The spinal cord receives its blood supply from the anterior and posterior spinal arteries, both of which arise from the vertebral, and from the intercostal branches. These intercostals have ascending and descending branches, which anastomose freely with one another and with the anterior and posterior spinal arteries, making an arterial network about the spinal cord, from which numerous small branches penetrate the surface. The gray matter receives its blood supply principally from the anterior spinal arteries, and it is to be noted that this supply is largely from different branches than that of the white matter. It is also to be remembered that the arteries which enter the cord are terminal. Considering this, it can be readily understood how a destruction of a number of spinal segments would cause interference with the blood supply, first of the whole cord, and after the acute symptoms have subsided, especially of the contiguous parts. This arterial obliterative process in common with all degenerations has a tendency to increase, and if the patient lives long enough, would involve considerable portions of the spinal cord.

In the case with necropsy (Case 3) the arterial degeneration is evident. Sections of all parts of the cord show marked endarteritis and this seems to be much more apparent in the lower parts of the cord near the lesion, and especially so in the periphery and in the lateral columns. The vessels in the gray matter seem less sclerosed, the reason for this greater endarteritis in the white matter of the cord being the more ready involvement of the arterial supply. This has only recently been called attention to by Allen.[1]

It is probable that a similar arteritic process is present in the clinical cases here recorded, but of much greater intensity as the lesions are of 35 and 23 years' duration as compared with 4 years in the case with pathological findings. This endarteritis causes impairment in the nutrition of the parts; this resulting in a gradual necrobiosis and impairment of function. In this is to be found the probable explanation of the increase in the sensory disturbances.

What influence, if any, do the secondary degenerations have? This question has long been in dispute, from the time that Charcot first explained the occurrence of the contractures in hemiplegia as the result of secondary degeneration of the motor columns. Take, for instance, in the case with necropsy. Here as a result of a myelitis in the 9th thoracic segment, marked secondary degenerations are to be found in the cervical cord. In studying such sections stained by the Weigert hematoxylin method it seems as if these extensive degenerations must have some influence, if not upon the neighboring fibers, at least upon the fibers which mingle with them.

Let us try to analyze this. In a lesion of one internal capsule interrupting the motor columns, secondary degeneration results throughout the whole extent of the pyramidal tract. Such degeneration will, at first, only be apparent by the Marchi and later by the Weigert hematoxylin method.

The question is whether this has any influence upon the structures which are associated with it. We assume that there is a connection between the cells of the anterior horns of the spinal cord and the motor columns, and if this is true, as we have every reason to believe, a degeneration of the motor columns should have influence upon these cells, and yet pathologically this has never been satisfactorily demonstrated. That is no argument, however, that this does not actually occur. Clinical evidence shows sufficiently that in hemiplegia there are atrophy and vasomotor disturbance; these have never been satisfactorily explained, but are probably due to a loss of tone of the whole motor system of which the motor nuclei in the anterior horns of the cord play a prominent part.

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[1] A. R. Allen, University of Pennsylvania Medical Bulletin, 1905.

When a paralysis occurs, as for instance, because of a capsular lesion, how is loss of power produced? There is as a result of this lesion, immediate disturbance of physiological relation between the cortical motor cells, the pyramidal tracts, the cells of the anterior horns, the anterior spinal roots, the peripheral nerves, and the muscles, tendons and fascia these supply. More than this, there must be some disturbance of the normal relation with the sensory arc.

This disturbance of physiological relation, or "diaschisis," as Von Monakow has termed it, is, of course, immediate, and will become permanent in varying degree; this depending upon the extent of the original lesion. And how is this disturbance of relation manifested? Clinically by loss of power and pathologically by degeneration of fibers. This degeneration, which is called secondary, is progressive as shown by the reaction to the Marchi and Weigert stains. So far our microscopic methods have failed to demonstrate any changes in the cells of the anterior horns, and in the nerves and muscles. This, however, is mainly because of the recuperative power possessed by the motor cells on account of the innervation received from other sources, and the independent trophic action which they possess. We see then that what is termed secondary degeneration is really nothing more than the physiological or mechanical death of a part.

What action, if any, then, has this secondary degeneration or physiological death of a part? Evidently none, for whatever has produced this, has at the same time produced loss of function in the related parts. Secondary degeneration, however, does produce from the very nature of its slow death, some physiological disturbance. Take for instance the motor columns. Even though there is a complete destruction of these as a result of a capsular lesion, other motor fibers, the so-called extra-pyramidal bundles, join the motor columns in their course downwards. Whether these bundles intermingle freely with the motor columns in the pons and medulla, or whether they are independent in their course, has not been fully determined; but it is known that they ultimately join the motor columns in the spinal cord and probably mingle with them. We can readily imagine that the degenerated or dead fibers must have some detrimental action upon the healthy fibers.

This can be better demonstrated in the so-called ascending or sensory degenerations. Take, for instance, in Case 3, in which because of a lesion in the 9th thoracic segment, the secondary degenerations are marked. As we go higher and higher, newer fibers enter by means of the posterior roots, and join either the posterior columns or the columns of Gowers and the direct cerebellar tracts. These newer fibers intermingle, or at least, accompany the degenerated fibers.

This influence is probably the result of a vascular change. The blood vessels that supply or are in association with a degenerating or degenerated tract must share somewhat in the sclerotic process, and it is conceivable that as a result of this obliterative arteritis, some degeneration is produced in the contiguous or intermingling healthy fibers.

In the case with necropsy a careful examination of sections of the spinal cord above the lesion showed that the fibers in the posterior columns and in the entrance root zones were much better or more deeply stained than the fibers in the other parts. The pallor in the centripetal columns can be explained as the result of secondary degenerations, but this does not explain the apparent degeneration in the motor columns. Retrograde atrophy of the pyramidal tracts has been described, and it occurs in the motor columns above destructive lesions of the spinal cord. This is as we should expect, for when physiological relations are disturbed, as they would be in such case, there must be some disturbance of function in the whole motor system as has been indicated above. This disturbance is manifested pathologically by a downward degeneration of the motor columns below the point of lesion, but is not apparent in most instances in the parts above, at least, by our pathological methods. This is because these fibers still retain their trophic cortical innervation. Reasoning from this then, retrograde atrophy must occur in every case, in the motor as well as the sensory tracts.

The increase in the sensory phenomena in the parts above the peripheral distribution related to definite lesions of the cord, has an important practical bearing. Most of our observations upon sensory areas are based on definite lesions of the spinal cord, in which no attention has been paid to a possible increase of the sensory phenomena, as is pointed out in this paper. Manifestly then such observations in which the lesions are of long duration cannot be as accurate as those which are the result of lesions of a shorter time. In this is to be found one explanation of the great variance in the sensory diagrams.

The second part of this paper is to again call attention to the increase of reflexes in the parts above definite lesions of the spinal cord. This clinical fact was first called attention to by me in 1904. ("Clinical Report of Three Cases of Injury to the Lower Spinal Cord and Cauda Equina."[2]) In this paper I called attention to the increase of patellar jerks in a lesion of the cauda equina. As this subject is an important one and as no other writer has contributed to it, some of the remarks then made pertaining to the subject will be quoted.

"I have been unable to find any reference to this condition in the literature at my command, except in Thorburn's[3] contribution to the surgery of the spinal cord. This author mentions the following cases with increase of the patellar reflexes, but pays no special attention to this subject. One case I have found also reported by Franz Volhard.[4]

"First case, cited by Thorburn from Kirchoff; backward crushing of the first lumbar vertebra causing a degeneration of the fourth and fifth sacral segments, the only symptoms being paralysis of the bladder and rectum and increased patellar reflexes. Sexual and sensory changes are not mentioned.

"Second case: a partial compression of the cauda equina about the level of the last lumbar vertebra, causing severe neuralgic pains in the sciatic and pudic distributions, a weakness of some of the muscles of the lower limbs, but no complete paralysis and no anesthesia of the limbs. The bladder and rectal functions were paralyzed. The patellar reflexes were slightly exaggerated, but there was no ankle clonus.

"The third case is cited by Thorburn from Oppenheim, and was one of fracture of the first lumbar vertebra, causing a myelitis of the conus medullaris. There was a slight weakness of the calf muscles, otherwise no loss of power or atrophy. Anesthesia was limited to the peroneal region and buttocks. There was paralysis of bladder and rectum. Here also the knee-jerks were exaggerated, but there was no ankle clonus.

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[2] The American Journal of the Medical Sciences, May, 1904.
[3] Philadelphia, Blakiston & Son, 1899.
[4] Deutsche med. Wochen., 1902, No. 33.

"The fourth case, by Volhard, of a tumor of the cauda equina, as proved by necropsy, with motor and sensory symptoms, absent Achilles jerks, and weakness of the plantar reflexes, but both the patellar reflexes and the cremasteric reflexes were exaggerated.

"In all four of these cases, therefore, the exaggeration of the patellar reflexes was probably caused by lesions below the reflex arc."

In the two clinical cases now recorded, the tendon reflexes in the upper limbs, that is, the biceps and triceps, were very prompt and the skin reflexes over the shoulders and chest were exceedingly exaggerated. Tapping over any portion of the chest, shoulders or back produced prompt and marked contraction in the corresponding reflex arcs. How is this to be explained? Quoting again:

"It is difficult to find a satisfactory explanation for such a phenomenon as this, but evidently the reflex arcs are in some way thrown into a state of excitation in these cases. No writer has made this a subject of careful study, and it seems therefore important to emphasize the fact that a reflex may be increased by a lesion in the spinal cord below the portion in which the reflex arc is represented. Considerable evidence is offered that in the nervous system there are both depressomotor and excitomotor fibers for the different reflexes, these having their origin in the brain; and it may be that other excitomotor fibers arise. We know that spinal roots on entering the spinal cord give off descending branches that pass downward in the posterior columns; the function of these fibers is entirely unknown, but we must assume that in some way they affect the function of the lower segments. It is probable that in a similar manner the lower spinal segments exert some influence over higher segments, and there is no doubt whatever that in the antero-lateral columns degeneration of short fibers occurs upward. It is presumable that these fibers exert some control over higher segments than those in which they arise, and in this way possibly cause an exaggeration of tendon reflexes."

It is also probable, as has been shown in the present paper, that the disturbance in the arterial supply in the parts above definite lesions of the cord, produces a disturbance in the functions in the motor columns, this being further influenced by the retrograde atrophy. As has been shown by Rothmann, the only constant symptom of a lesion of the motor column is an exaggeration of the reflexes. Here, then, is to be found the explanation of the reflexes in the parts above lesions of the spinal cord.

The following conclusions can be drawn from the study of this paper:

First: An injury to the spinal cord will cause at first interference with the blood supply of the whole cord. If, as a result of such an injury, there should be produced a limited lesion of the spinal cord, there will be interference with the blood supply of the contiguous areas. This interference will be progressive, thus causing arterial obliteration and necrosis in the contiguous parts.

Second: This progressive degeneration will cause disturbance of function, it being manifested clinically by a gradual extension of the areas of disturbed sensation, and by increase in the tendon and skin reflexes.

Third: A lesion in any portion of the spinal cord will cause disturbance of physiological relations in the associated parts. This disturbance is greatest directly after the injury, and becomes less in the course of time.

Fourth: Secondary degenerations "per se" do not produce any direct symptoms, for whatever has produced secondary degenerations, has at the same time caused loss of function in the related parts. Secondary degenerations, however, cause some physiological disturbance. Every degenerating or degenerated tract has healthy fibers from other sources mingling with it. The arterial degeneration present in the involved tract will cause degeneration in these healthy fibers, and of the immediate fibers surrounding the degenerating tract.